

## Splenic granuloma: Melioidosis or Tuberculosis?

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### ABSTRACT

Melioidosis well known as a 'great mimicker' is caused by *Burkholderia pseudomallei*. Even though majority of the patients present with acute infection, around 18 % can present as chronic infection. These latent foci of infection may reactivate to cause fulminant infection at a later date. Due to lack of clinical suspicion and good laboratory facility latent infections are often misdiagnosed and treated as tuberculosis. Chronic splenic granuloma is a rare manifestation of Melioidosis . Deep seated abscess requires atleast 4 weeks of intensive treatment with intravenous antibiotics. Ceftazidime , the drug of choice for melioidosis can cause drug induced thrombocytopenia. Simultaneous use of diclofenac may potentiate this phenomenon. Treatment with meropenem may be life saving in such situations.

**Keywords:** Drug-induced thrombocytopenia, melioidosis, splenic granuloma, tuberculosis

### Introduction

Melioidosis is caused by a soil saprophyte *Burkholderia pseudomallei*. The clinical manifestations of melioidosis can range from pneumonia to pericardial effusion and multiple visceral abscesses and hence have been appropriately termed as "mimicker of maladies."<sup>[1,2]</sup> When majority of patients present with acute infection, 18% of the patients present with chronic infection with symptoms for more than 3 months. Analogous to tuberculosis (TB), *B. Pseudomallei*, can remain latent for decades and then reactivate.<sup>[3]</sup> Growth in culture media is the gold standard for the diagnosis of melioidosis. Due to lack of physicians' awareness and lack of good quality laboratory facilities, erratic diagnosis and treatment are common.<sup>[4]</sup>

We report a case of chronic melioidosis in a middle-aged gentleman who presented with pyrexia of unknown origin and had sonological features suggestive of splenic granuloma.

### Case Report

A 53-year-old farmer known to have diabetes mellitus with well-controlled blood sugars presented with intermittent episodes of high grade fever for more than a year. He had occasional dull aching pain in the left subcostal region. There was no history of reduction of weight or appetite. He never consumed ethanol. In the beginning, he received a course of antitubercular drugs for 6 months duration for a diagnosis of extrapulmonary TB in spleen, despite which his symptoms persisted.

On examination, he had normal vital signs and insignificant axillary lymphadenopathy. He had mild hepatosplenomegaly and a dull Traube's space. Rest of his systemic examination was unremarkable. The differentials considered for hypoechoic lesions in the spleen with pyrexia of unknown origin were extrapulmonary TB, sarcoidosis, infective endocarditis, splenic marginal zone lymphoma, *B. pseudomallei* abscess, histoplasmosis, and hematological malignancy.

His routine blood investigations were normal except for an elevated C-reactive protein (CRP) of 9.8 mg/L (ref range: <6 mg/L) and erythrocyte sedimentation rate of

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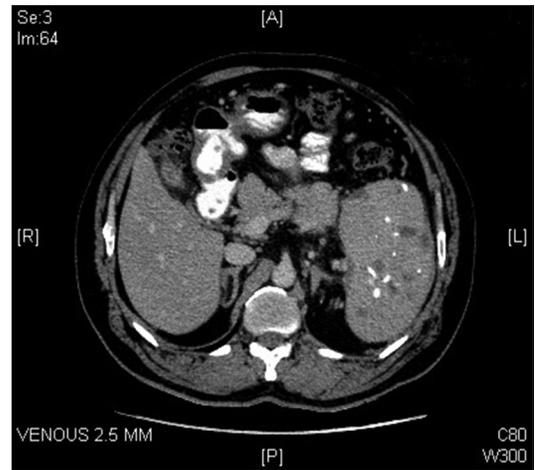
60 mm/h (ref range: 3–10 mm/h). At admission, his glycated hemoglobin was 6.7%. Sonography of the abdomen revealed multiple hypoechoic lesions in the spleen with peripheral calcification which was suggestive of multiple granulomas. These findings were confirmed on computed tomography scan of the abdomen [Figures 1-3]. Serial blood cultures showed no growth. Blood-borne virus screen, angiotensin-converting enzyme levels, chest X-ray, echocardiography, and bone marrow examination were normal. Ultrasound-guided fine-needle aspiration cytology of the splenic lesion was negative for X-pert TB polymerase chain reaction and for malignant cells. Axillary lymph node biopsy did not yield any positive results. Diagnostic splenectomy was being considered when bacterial culture from the splenic aspirate grew *B. pseudomallei* and he was initiated on intravenous ceftazidime.

Three days later, he was noted to have icterus. Liver function tests revealed indirect hyperbilirubinemia (total bilirubin 2.2 mg/dL [ref range: 0.5–1 mg/dL]) and direct bilirubin 1.7 mg/dL with normal liver enzymes. His platelet counts dropped to  $<15,000/\text{cumm}^3$ . He had mucosal bleeding and melaena necessitating platelet transfusions. The differentials considered for his thrombocytopenia were DIC, TTP, and drug-induced thrombocytopenia. However, his fibrinogen level was normal, schistocytes were absent, and coagulation parameters were normal. It was found that he was on diclofenac for pain relief after lymph node biopsy and it was stopped. Thrombocytopenia persisted despite stopping diclofenac, and ceftazidime-induced immune thrombocytopenia was considered. His antibiotic therapy was changed to meropenem along with 4 days of dexamethasone. He received 4 weeks of intravenous antibiotics and was discharged with an advice to continue co-trimoxazole and doxycycline for a period of 3 months. His platelet counts improved, and he was well at the time of discharge. During the follow-up visits, his CRP showed declining trend, and he was found to be well.

## Discussion

Melioidosis is an emerging disease in India. Male sex, diabetes mellitus, chronic steroid use, and chronic alcohol intake are predisposing factors for melioidosis. However, around 16% of the patients do not have any risk factors.<sup>[1,4]</sup> Even though our patient had diabetes mellitus, his glycaemic control at the time of presentation was normal. Acquiring of his infection could be attributed to the time when he had poor glycaemic control.

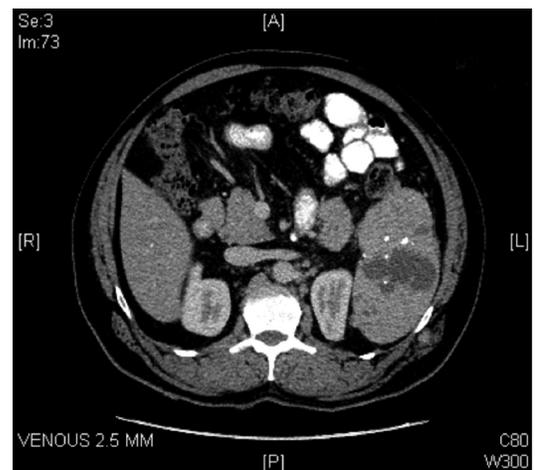
Melioidosis can present with vivid clinical manifestations ranging from latent melioidosis, intraabdominal abscesses, and chronic melioidosis to acute fulminant forms which may be fatal. Chow *et al.* recently reported asymptomatic latent melioidosis diagnosed from the posttraumatic splenectomy specimen. The surgical specimen biopsy showed typical granuloma which could not be differentiated from TB based on histopathology.<sup>[5]</sup> Chronic melioidosis may appear as granulomas on imaging and histopathology and can mimic



**Figure 1:** Computed tomography abdomen showing splenic lesions with peripheral calcifications



**Figure 2:** Computed tomography abdomen axial cuts with hypodense lesions in spleen



**Figure 3:** Computed tomography abdomen with multiple hypodense lesions in spleen

other granulomatous diseases. This reiterates the importance of culture-based diagnosis.

*B. pseudomallei* is a slow-growing bacterium which requires a selective medium for isolation from nonsterile sites. Laboratory diagnosis of *Burkholderia* includes three steps - initial screening followed by phenotypic and genotypic tests which are not available in most of the laboratories.<sup>[6]</sup> Due to similarity in phenotypic characters, *Burkholderia* can be mistaken for pseudomonas in inexperienced hands.<sup>[7]</sup> High clinical suspicion and good laboratory are imperative for the diagnosis of melioidosis.

Cephalosporins and diclofenac can cause immune thrombocytopenia. Cephalosporins act as haptens and attach to the platelet membrane and cause immune reaction and thrombocytopenia.<sup>[8]</sup> It is not known whether combining both the drugs potentiates this phenomenon. The platelet counts normalized after stopping the offending drugs and treatment with dexamethasone in our patient.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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